

(FILE 'HOME' ENTERED AT 17:20:16 ON 13 FEB 2000)

FILE 'MEDLINE, EMBASE, SCISEARCH, BIOSIS, USPATFULL' ENTERED AT 17:20:57
ON 13 FEB 2000

L1	0 S LIPASE? SAME ANTIGEN?
L2	2122 S LIPASE? AND ANTIGEN?
L3	425 S ANTILIPASE OR ANTI-LIPASE OR (ANTIBOD? (2A) LIPASE?)
L4	92 S L2 AND L3
L5	737547 S TRIACYLGLYCEROL? OR LIPID?
L6	40896 S FATTY ACID? AND GLYCEROL?
L7	13137 S L5 AND L6
L8	1 S L7 AND L4
L9	21 S L7 AND L3
L10	28239 S L5 AND LIPASE?
L11	118 S L10 AND L3
L12	502 S L7 AND (AVIAN OR CHICKEN OR POULTRY)
L13	0 S L12 AND L3
L14	255 S L12 AND (FOOD OR FEED)
L15	50 S L14 AND LIPASE?

L11 ANSWER 1 OF 118 MEDLINE

ACCESSION NUMBER: 1999443382 MEDLINE

DOCUMENT NUMBER: 99443382

TITLE: Lipoprotein hydrolysis and fat accumulation in chicken adipose tissues are reduced by chronic administration of lipoprotein **lipase** monoclonal **antibodies**

AUTHOR: Sato K; Akiba Y; Chida Y; Takahashi K

CORPORATE SOURCE: Department of Animal Science, Faculty of Agriculture, Tohoku University, Sendai-shi, Japan..
kan@bios.tohoku.ac.jp

SOURCE: POULTRY SCIENCE, (1999 Sep) 78 (9) 1286-91.
Journal code: PG3. ISSN: 0032-5791.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200001

ENTRY WEEK: 20000104

AB The lipoprotein **lipase** (LPL) catalyzed hydrolysis of plasma lipoproteins is a rate-limiting step in the **lipid** transport into peripheral tissues. The aim of the present study was to isolate monoclonal

antibodies against chicken adipose LPL and to investigate whether chronic infusion of the LPL monoclonal antibodies inhibits adipose LPL activity and consequently reduces fat accumulation in broiler chickens. The LPL catalyzed very low density lipoprotein (VLDL) hydrolysis was completely inhibited by the addition of 100 microg/mL of monoclonal antibodies (CLP10, CLP14, CLP16) in the in vitro incubation with plasma VLDL and

LPL.

A single injection of CLP10 and CLP16 into chickens fed or starved for 24 h elevated plasma **triacylglycerol** concentrations for 24 h, whereas that of CLP14 was ineffective. Intravenous injection every other day and continuous infusion by osmotic minipump with CLP16 maintained higher plasma **triacylglycerol** concentration for 5 d than that of the control group and extensively reduced LPL activity in adipose tissues and abdominal fat pad weight. Lipoprotein **lipase** mRNA and protein levels in adipose tissue were not modified by chronic administration of anti-LPL antibody. The results indicate that chronic administration of anti-LPL antibodies is effective in retarding fatness

in

broiler chickens, and the antibodies are a proper subject for studies of lipoprotein metabolism.

L11 ANSWER 8 OF 118 MEDLINE

ACCESSION NUMBER: 91182280 MEDLINE

DOCUMENT NUMBER: 91182280

TITLE: Metabolism of very low density lipoproteins in genetically lean or fat lines of chicken.

AUTHOR: Leclercq B; Hermier D; Guy G

CORPORATE SOURCE: INRA, Station de Recherches Avicoles, Nouzilly, France.

SOURCE: REPRODUCTION, NUTRITION, DEVELOPMENT, (1990) 30 (6)
701-15.

Journal code: AEW. ISSN: 0181-1916.

PUB. COUNTRY: France
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199107

AB Metabolism of very low density lipoproteins (VLDL) has been compared in fat (FL) and lean (LL) lines of chicken. When refed after fasting, plasma triglyceride concentration reached a significantly higher plateau in FL, although their feed consumption was lower than in LL. Newly synthesized VLDL were studied using anti-lipoprotein **lipase antibodies**. VLDL triglyceride (TG) concentrations were increased by antibody injection and reached a higher concentration in FL plasma

than

in LL. Newly synthesized VLDL exhibited a similar **lipid** composition. Fatty acid profiles were also similar when birds ingested a very low fat diet. Comparison of in vitro affinity of lipoprotein **lipase** and VLDL from both genotypes did not reveal any difference in Km and Vmax. [14C]labelled VLDL from fat or lean donors were prepared and were injected into chickens from both genotypes. Fractional rate constants did not differ between lines. However, as plasma VLDL-TG pools were very different, plasma turnover was higher in FL than in LL. About 3-fold more VLDL-TG were incorporated in abdominal fat of FL than in LL. Difference in fattening between both genotypes seem to be due to both increased VLDL secretion and VLDL removal from plasma without difference in VLDL characteristics.

L11 ANSWER 21 OF 118 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 1999005117 EMBASE

TITLE: Detailed characterization of the binding site of the lipoprotein **lipase**- specific monoclonal **antibody** 5D2.

AUTHOR: Chang S.-F.; Reich B.; Brunzell J.D.; Will H.

CORPORATE SOURCE: H. Will, Heinrich-Pette-Institut, Experimentelle Virologie/Immunologie, Universitat Hamburg, Martinistrasse 52, 20251 Hamburg, Germany

SOURCE: Journal of Lipid Research, (1998) 39/12 (2350-2359).

Refs: 43

ISSN: 0022-2275 CODEN: JLPRAW

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Monoclonal antibody (MAb) 5D2 recognizes lipoprotein **lipases** (LPL) from different species but not related **lipases**. This MAb is a unique reagent, used world-wide, because it differentiates between monomeric inactive and dimeric active LPL, inhibits human LPL enzyme activity, and binds to C-terminal LPL sequences involved in interactions with lipoproteins, lipoprotein receptors, and heparin. In this study we have analyzed the fine specificity of the MAb epitope recognition in

order

to better understand its functional properties and species-specific LPL immune reactivity. In peptide scan assays, MAb 5D2 reacted with all, except two, 13 amino acid-long peptides located between positions 380 and 410. Peptides from the amino terminal end of this region reacted more strongly than those from the carboxyl terminal end. Furthermore, only a peptide from the amino terminal end competed effectively with the binding of MAb 5D2 to native LPL bound to microtiter plates or nitrocellulose. A systematic peptide mutagenesis study indicated that 8 amino acids of the reactive region, mainly located in the amino terminal end, are critical for binding and probably directly interact with MAb 5D2. The experimentally determined antigenicities of species-specific LPL peptides and of the corresponding denatured full-length LPL proteins on

immunoblots

were consistent with these findings. According to a proposed 3D-model for LPL, only the amino terminal end of the antigenic region is easily surface-accessible. These data combined with 3D-modelling of monoclonal **antibody** (MAb)-lipoprotein **lipase** (LPL) protein interaction provide new insight into the known biological effects of MAb 5D2 on LPL and the antigenic determinants that are recognized.

FILE 'MEDLINE, EMBASE, SCISEARCH, BIOSIS' ENTERED AT 15:58:56 ON 13 FEB 2000

L1 3166 S ENCAPSULAT? (5P) (IMMUNOGLOBULIN? OR ANTIBOD?)
L2 418 S (ANTIBOD? (3A) LIPASE?) OR ANTILIPASE? OR (ANTI LIPASE?)
L3 0 S L1 AND L2
L4 44 S (MAMMAL OR AVIAN) AND L1
L5 0 S (WEIGHT (2A) (CONTROL? OR REGULAT?)) AND L2
L6 0 S (WEIGHT (2A) (CONTROL? OR REGULAT?)) AND L4
L7 0 S L4 AND LIPASE
L8 161 S (WEIGHT (2A) (CONTROL? OR REGULAT?)) AND LIPASE
L9 0 S L8 AND L4
L10 0 S L8 AND L2
L11 0 S L8 AND L1

(FILE 'HOME' ENTERED AT 15:58:11 ON 13 FEB 2000)

FILE 'MEDLINE, EMBASE, SCISEARCH, BIOSIS' ENTERED AT 15:58:56 ON 13 FEB 2000

L1	3166 S	ENCAPSULAT? (5P) (IMMUNOGLOBULIN? OR ANTIBOD?)
L2	418 S	(ANTIBOD? (3A) LIPASE?) OR ANTILIPASE? OR (ANTI LIPASE?)
L3	0 S	L1 AND L2
L4	44 S	(MAMMAL OR AVIAN) AND L1
L5	0 S	(WEIGHT (2A) (CONTROL? OR REGULAT?)) AND L2
L6	0 S	(WEIGHT (2A) (CONTROL? OR REGULAT?)) AND L4
L7	0 S	L4 AND LIPASE
L8	161 S	(WEIGHT (2A) (CONTROL? OR REGULAT?)) AND LIPASE
L9	0 S	L8 AND L4
L10	0 S	L8 AND L2
L11	0 S	L8 AND L1

FILE 'MEDLINE, EMBASE, SCISEARCH, BIOSIS, USPATFULL' ENTERED AT 16:11:14 ON 13 FEB 2000

L12	6106 S	ENCAPSULAT? (5P) (IMMUNOGLOBULIN? OR ANTIBOD?)
L13	560 S	(ANTIBOD? (3A) LIPASE?) OR ANTILIPASE? OR (ANTI LIPASE?)
L14	3 S	L12 AND L13
L15	327 S	(WEIGHT (2A) (CONTROL? OR REGULAT?)) AND LIPASE
L16	8 S	L15 AND L1